Pattern of focal γ-bursts in chess players

Grandmasters call on regions of the brain not used so much by less skilled amateurs.

The brain's medial temporal lobe structures are thought to be important for the initial formation of long-term memory[1,2], and active memory is indicated by bursts of γ-band activity in these and other areas of the association cortex[3,4]. Here we use a new technique of magnetic imaging to compare focal bursts of γ-band activity in amateur and professional chess players during matches. We find that this activity is most evident in the medial temporal lobe in amateur players, which is consistent with the interpretation that their mental acuity is focused on analysing unusual new moves during the game. In contrast, highly skilled chess grandmasters have more γ-bursts in the frontal and parietal cortices, indicating that they are retrieving chunks from expert memory by recruiting circuits outside the medial temporal lobe.

The 'chunking' theory of chess playing[5] suggests that expert memory is based on a large database of chunks in long-term memory. A chess grandmaster studies and practises for at least 10 years to learn more than 100,000 patterns (memory chunks). Consequently, grandmasters can 'recognize' the key elements in a problem situation much more rapidly than amateur players. Experts differ not only in the extent of their knowledge, but also in its organization. High-level processing elements, such as structuring knowledge and planning, assist in accessing the respective chunks[6].

We tested 20 male players (aged 42 ± 14 years), each with more than 10 years of tournament and training practice. Ten professional grandmasters scored between 2,400 and 2,600 on Elo's chess-skill rating scale[5]; amateur players ranked 1,700 and above. Magnetoencephalographic recordings were made while subjects played against a computer and then were scanned in the 5 seconds after each move by the computer program for focal γ-bursts (20–40 Hz; Fig. 1a). Examination of single slices indicates pronounced activity in the region of the perirhinal and entorhinal cortex, hippocampus and related structures in amateur players, but not in grandmasters.

There was a strong negative correlation ($r = -0.84$) between the relative share of dipoles in these structures and Elo chess skill (Fig. 1b). The correlation was also significant when only the 12 players who had lost in the game were included in the analysis (draws and wins were achieved only by grandmasters). There was no relationship between the length or complexity of the game and the Elo score.

These marked differences in the distribution of focal brain activity during chess playing point to differences in the mechanisms of brain processing and functional brain organization between grandmasters and amateurs. Lesions in structures that are activated in amateur players impair recent memory while leaving remote memory intact[2]. Grandmasters seem to rely more on remote than on recent memory.

High-level processing elements[6] may also have contributed to the observed differences. The chunking theory of memory states that the number and nature of chunks that chess experts can hold in long-term memory can be used to predict chess performance[6]. Our results indicate that the activation of expert memory chunks produces focal γ-bursts in the neocortex, whereas amateur players primarily encode and analyse new information, tasks that activate the medial temporal lobe and the hippocampus. It is possible that these structures play only a transitional role during the establishment of expert memory in the neocortex.

Ognjen Amidzic, Hartmut J. Riehle,
Thorsten Fehr, Christian Wienbruch,
Thomas Elbert
University of Konstanz, Universitätstrasse 10,
Fach-D30, 78457 Konstanz, Germany
e-mail: ognjen.amidzic@uni-konstanz.de

1. Bontempi, B., Laurent-Demir, C., Destrade, C. & Jaffard, R. Nature 400, 671–674 (1999).
2. Squire, L. R. Psychol. Rev. 99, 195–231 (1992).
3. Pulvermüller, F., Keil, A. & Elbert, T. Trends Cogn. Sci. 3, 250–252 (1999).
4. Singer, W. et al. Trends Cogn. Sci. 1, 252–261 (1997).
5. Elo, A. E. The Rating of Chess Players, Past and Present (Arco, New York, 1978).
6. Gobet, F. Cognition 66, 115–152 (1998).
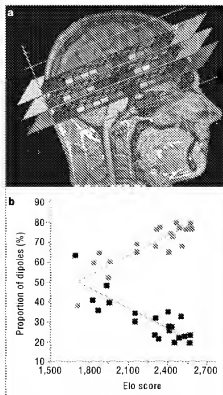
Figure 1 γ-band activity in the brains of chess players. a, Determination of equivalent-current dipole density for inferior 'slices' through the hippocampus and medial temporal areas in an amateur player's brain. Increasing dipole density is indicated by a colour scale from dark to light blue, to yellow, to red. b, Relationship between chess-playing skill (Elo rating scale) and the relative share of dipoles located in medial temporal lobe structures (black) and in the frontal and parietal cortices (red). Amateur players show more focal γ-bursts in the medial temporal lobe than grandmasters, who show more activity in the frontal and parietal cortices.

Low mechanical signals strengthen long bones

Although the skeleton's adaptability to load-bearing has been recognized for over a century[1], the specific mechanical components responsible for strengthening it have not been identified. Here we show that after mechanically stimulating the hindlimbs of adult sheep on a daily basis for a year with 20-minute bursts of very-low-magnitude, high-frequency vibration, the density of the spongy (trabecular) bone in the proximal femur is significantly increased (by 34.2%) compared to controls. As the strain levels generated by this treatment are three orders of magnitude below those that damage bone tissue, this

anabolic, non-invasive stimulus may have potential for treating skeletal conditions such as osteoporosis.

A common perception of bone adaptation is that mechanical signals must be large to influence morphology[2]. The peak signals that result from natural vigorous activity cause microdamage to bone material and require repair[3]. For example, peak strains of 2,000–3,000 microstrain are typically induced during locomotion[4], stimulating osteoclasts and osteoblasts to remove and then replace damaged tissue[5].

We have departed from this repair-mediated hypothesis by proposing that extremely small strains (for example, those that arise from muscle contraction during less vigorous but more frequent activities such as maintaining posture) are strong determinants of bone morphology[6].

We examined the regulatory potential of extremely small (0.3g, where g is the Earth's gravitational field), high-frequency (30 Hz) mechanical accelerations by subjecting the hindlimbs of adult (6–8-year-old) female sheep (Warhill, intact ewes) to a ground-based vertical oscillation[7] for 20 min per day for 5 days a week. When the animals were not being treated, they joined the controls to roam freely over a pasture area. We used strain gauges attached to the animals' tibia bone to calibrate the device: these showed that the peak-to-peak amplitude of the strain generated was about 5 microstrain, which is 0.1% of the strain magnitude that is known to cause yield-failure in bone[8].

After 1 year of this mechanical-stimulation regime, the density of trabecular bone in the proximal femur, as quantified by computer tomography, was 34.2% greater in experimental sheep than in controls ($P < 0.01$; Table 1). This strong anabolic response was substantiated by undecalcified bone histology of the same region, which revealed a 32% increase in trabecular bone volume, a 45% increase in trabecular mesh number (Fig. 1) and a 36% reduction in mesh spacing, indicating an increase in the mean width of each trabecular element and the addition of new trabeculae.
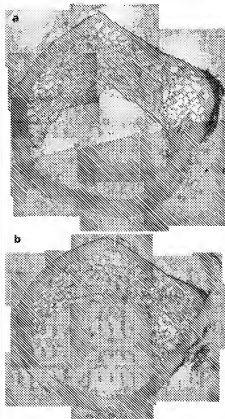
Figure 1 Montages of photomicrographs of the proximal sheep femur after 1 year of exposure (20 min per day) to a 0.3g, 30-Hz mechanical stimulus. a, b, There is 32% more trabecular bone in the proximal femur of experimental animals (a) as compared with age-matched controls (b) ($P < 0.04$).

Table 1 Proximal-femur parameters of control and stimulated sheep

	Control	Experimental	Difference	P
Animal mass (kg)	71.1 ± 7.1	70.3 ± 9.4	− 1.1%	n.s.
Total density (gm cm⁻³)	466 ± 60	496 ± 53	+6.5%	< 0.1
Trabecular density (gm cm⁻³)	169 ± 37	227 ± 56	+34.2%	< 0.01
Bone volume/total volume (%)	15.2 ± 4.1	20.1 ± 4.8	+32%	< 0.04
Trabecular spacing (μm)	1,170 ± 124	756 ± 97	− 36%	< 0.02
Trabecular number (trabeculae mm⁻¹)	0.82 ± 0.16	1.19 ± 0.18	+45%	< 0.01
Bone-formation rate (μm³ mm⁻¹)	8.4 ± 12.7	17.9 ± 16.3	+113%	< 0.2
Mineralizing surface (%)	2.6 ± 0.16	6.34 ± 5.14	+144%	< 0.1

Animal mass and envelope-specific bone density (determined by quantitative computer tomography) of the proximal femur after 12 months of low-level mechanical stimulation. Also shown are indices of static and dynamic histomorphometry of the proximal femur. Although 'whole-bone' parameters of the proximal femur show only a limited tendency to be enhanced by mechanical stimuli ($P \sim 0.1$), this increase in treated animals compared with controls is over 30% for trabecular bone alone ($P < 0.01$). One control was lost over the course of study for reasons not associated with this protocol. One animal was evaluated in the experimental group, with eight controls. All evaluations were made without knowledge of whether the animals were control or experimental

We found that this low-level mechanical stimulation increased the rate of bone formation 2.1-fold ($P < 0.2$) and the mineralizing surface 2.4-fold ($P < 0.1$). This anabolic effect was highly specific to cancellous (porous) bone, as there was no significant histomorphometric change in any of the cortical bone parameters. We detected no difference in any bone index in the radius of either control or experimental animals (for example, mineral density was 0.6% less than controls: not statistically significant), indicating that the anabolic effect was specific to the region of the skeleton that was subjected to the mechanical signal.

Mechanical strain in the skeleton is a product of functional load-bearing — as seen, for example, in the mandible of the macaque[9] and the tibia of the alligator[10]. In addition to the large-amplitude strains typically associated with functional activity, a strain signal, much less than 5 microstrain in amplitude, arises through muscular activity in the frequency band 10–50 Hz (ref. 11). Generation of this small-amplitude, high-frequency muscle 'vibration' persists through even such passive activities as standing.

Skeletal morphology may therefore be sculpted by omnipresent, low-level muscle activity as well as by the peak impacts inherent in load-bearing. In terms of clinical relevance, the strong bone-generating capacity of these small signals suggests that biomechanical intervention might help to strengthen bone in osteoporosis sufferers without the side-effects associated with pharmacological treatment. In addition to being non-invasive and inducing a therapeutic response from the bone tissue itself, low-intensity mechanical signals incorporate all aspects of a complex remodelling cycle[12] and ultimately stimulate formation of lamellar bone[13] to improve bone quantity and quality.
Clinton Rubin*, A. Simon Turner†,
Steven Bain‡, Craig Mallinckrodt†,
Kenneth McLeod*

*Musculo-Skeletal Research Laboratory,
Department of Biomedical Engineering,
State University of New York, Stony Brook,
New York 11794–2580, USA
e-mail: clinton.rubin@sunysb.edu
†Department of Clinical Sciences, Colorado State University, Fort Collins, Colorado 80523, USA
‡Skeletech Inc., Bothell, Washington 98021, USA

1. Wolff, J. The Law of Bone Remodeling (transl. Maquet, P. & Furlong, R.) (Springer, Berlin, 1986).
2. Frost, H. Anat. Rec. 226, 403–413 (1990).
3. Burr, D. Martin, R., Schaffler, M. & Radin, E. J. Biomech. 18, 189–200 (1985).
4. Rubin, C. & Lanyon, L. J. Exp. Biol. 101, 187–211 (1982).
5. Schaffler, M., Radin, E. & Burr, D. Bone 11, 321–326 (1990).
6. Huang, R., McLeod, K. & Rubin, C. J. Gerontol. 54, 252–257 (1999).
7. Fritton, J., Rubin, C., Qin, Y. & McLeod, K. Ann. Biomed. Eng. 25, 831–839 (1997).
8. Carter, D., Harris, W., Vasu, R. & Caler, W. Am. Soc. Mech. Eng. 45, 81–95 (1981).
9. Hylander, W., Ravosa, M., Ross, C. & Johnson, K. Am. J. Phys. Anthropol. 107, 257–271 (1998).
10. Blob, R. & Biewener, A. J. Exp. Biol. 202, 1023–1046 (1999).
11. Fritton, S., McLeod, K. & Rubin, C. J. Biomech. 33, 317–326 (2000).
12. Bain, S. & Rubin, C. J. Bone Miner. Res. 5, 1069–1075 (1990).
13. Rubin, C., Gross, T., McLeod, K. & Bain, S. J. Bone Miner. Res. 10, 488–495 (1995).

vision

Realignment of cones after cataract removal

Through unique observations of an adult case of bilateral congenital cataract removal[1], we have found evidence that retinal photoreceptors will swiftly realign towards the brightest regions in the pupils of the eye. Cones may be phototropic, actively orientating themselves towards light like sunflowers in a field.

For 40 years, one of us (P.D.) lived with an unusual pupil configuration caused by congenital bilateral cataracts, first diagnosed at three years of age. The cataracts were managed by using a twice-weekly application of atropine, which dilated P.D.'s pupils to produce roughly annular (ring-shaped), clear regions around the dense nuclear cataracts. Because his optics were so poor (he was far-sighted, astigmatic and incapable of accommodation), P.D. squinted continuously as an adaptation, particularly for close work. His eyelids thus horizontally cropped each annulus at the top and bottom, leaving two clear entry points for light